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Lung Health Professional™
MAGAZINE

New Open Access Journal
Details on Page 15

COPD FOUNDATION

Feature Story:
COPD Foundation: A Decade of Progress
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Letter from the Managing Editor:

Katelyn Turner

2014 is an exciting year for the COPD Foundation, because we are celebrating our 10-year anniversary! Throughout the year, we will be featuring articles that commemorate the past decade of progress for the COPD Foundation and COPD community in general, denoting specific articles with our 10th anniversary logo. On behalf of the COPD Foundation, I want to thank all of our authors who make *Lung Health Professional* possible, and I want to thank all of you for your support. I encourage you to share COPD Foundation resources with your colleagues and patients—we are continuously receiving wonderful articles and are grateful to all who make it happen.

New COPD Journal:

We are excited to have another article that focuses on our open access journal in the field of COPD. *Chronic Obstructive Pulmonary Diseases: Journal of the COPD Foundation* will provide researchers and readers access to articles that their libraries do not subscribe to. We're proud to have an editorial board featuring leaders from across the globe who are all leaders in the field of COPD. You can find this article on Page 15.

COPD8USA Enduring Material:

The COPD Foundation, along with the COPD8USA Scientific Planning Committee Members, is pleased to provide you with an opportunity to receive continuing medical education credits. Please read more about this exciting opportunity on Page 26.

Feature on a Decade of Progress:

I am excited about our feature article, which was written by COPD Foundation President and Co-Founder John W. Walsh, COPDF Board Member Dr. Gerard Turino, COPDF Director of Education Scott Cerreta BS, RRT, and Patricia A. Jellen, RN. They all contributed their thoughts about the past decade of COPD from their own perspective, giving us an inside view to the COPD community.

Lung Cancer Screening and Pulmonary Rehab:

I am so grateful to Dr. Amir Sharafkhaneh and Dr. Richard Casaburi for submitting articles on lung cancer screening over the past decade (Page 22), and 10 years of stages of pulmonary rehab (Page 8), respectively. They are important articles, and fit well into our conversation of our 10-year anniversary.

As always, please feel free to send me your suggestions for topics for future issues of *LHP*. I welcome your input.

Please send suggestions to:
kturner@copdfoundation.org

Lung Health Professional magazine is published 4 times annually and is available from the COPD Foundation free of charge. If you would like to be added as a subscriber, please email Katelyn Turner at kturner@copdfoundation.org or call the C.O.P.D. Information Line 1-866-316-COPD (2673).

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References: 1. Data on file, PROLASTIN DIRECT program. **2.** Campos MA, Alazemi S, Zhang G, Wanner A, Sandhaus RA. Effects of a disease management program in individuals with alpha-1 antitrypsin deficiency. *COPD.* 2009;6:31-40. **3.** Data on file, Grifols.

Please see brief summary of PROLASTIN-C (alpha$_1$-proteinase inhibitor [human]) full Prescribing Information on adjacent page.

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IgA deficient patients with antibodies against IgA.

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- IgA deficient patients with antibodies against IgA are at greater risk of developing severe hypersensitivity and anaphylactic reactions.
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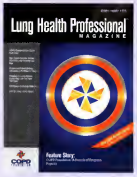

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COPD FOUNDATION

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COPD READMISSIONS SUMMIT

Integrating COPD into Patient Centered Hospital Readmission Reduction Programs

On October 11, 2013 the COPD Foundation convened national health service research experts, policy makers and foremost experts in COPD clinical care to help hospital systems, integrated delivery systems, academic centers and all those responsible for designing care models and programs for reducing readmissions answer challenging questions and ultimately improve outcomes for those living with COPD

Chronic Obstructive Pulmonary Disease (COPD) is an umbrella term used to describe progressive lung diseases including emphysema, chronic bronchitis, refractory (non-reversible) asthma, and some forms of bronchiectasis. This disease is characterized by increasing breathlessness

Hospital Readmissions Reduction Program

- Requires CMS to reduce IPPS reimbursements to hospitals with excess readmissions
- Since becoming effective on October 1, 2012 it has applied to three conditions
- COPD will be included in program beginning in FY15. A patient centered system for reducing hospital readmissions must account for the needs of the patient with COPD, without ignoring the reality of a complex patient and the demands of today's delivery environment.
- Interventions designed to reduce the risk of subsequent hospitalizations in patients recently hospitalized for COPD may have benefit, no effect, or harm.
- As a community, we need to take all possible measures ensure that no additional harm is caused.

Featuring

TRANSITIONAL CARE MODEL

BOOST

The Care Transitions Program

SUMMIT OVERVIEW

The summit was attended by roughly 100 in-person attendees and over 125 participants watching via live videostream.

- The summit kicked off with the first panel, chaired by Tom Kallstrom, RRT, presenting an overview of current COPD measures, hospital readmission statistics, major determinants of COPD related to 30-Day hospital readmissions, and research and demonstration projects addressing COPD.
- The second panel, moderated by Dr. Jill Ohar, included presentations of The Transitional Care Model, The Care Transitions initiative and Project BOOST.
- The third panel, moderated by Dr. Jerry Krishnan and John Walsh, included a discussion on future collaboration to address COPD related readmission reduction research and information dissemination.

REGISTERED IN-PERSON ATTENDEES	
Physician	20
nurse	18
Pharmaceutical Company	18
Repiratory Therapist	17
DME/Manufacturer	9
Healthcare Provider General	9
Hospital Adminsitrator	6
Patient	4
Insurer	2
Total	103

MOVING FORWARD

With all stakeholder groups present, the final session included a rich final discussion. Four themes emerged as a framework for moving forward:

1. COPD cannot be ignored in programs designed to improve post-discharge outcomes
2. 30-day Plus (30 day readmissions represent only one of several important outcomes; other outcomes include patient experience, functional capacity, and mortality)
3. Interventions to improve outcomes need to be tailored to the setting in which it is to be applied, Community Building and Patient Voice is needed to ensure that patient-centered interventions, rather than disease-specific strategies, are developed, tested, and implemented to improve outcomes important to patients.
4. An infrastructure to promote and sustain collaborations between patients, caregivers, providers, payers, researchers, and other stakeholders is needed to identify strategies of care needed to improve post-discharge outcomes in patients hospitalized for COPD exacerbations.

The stakeholders expressed interest in several specific outcomes

- Creating a common repository for current programs and knowledge to enhance community learning and urged the COPD Foundation to lead the way by creating a platform for collaboration
- A desire for a risk stratification tool for in-hospital and pulmonary rehabilitation use, and
- Future collaboration between stakeholders to share information including a webinar series and future summits. Individuals implementing different models should be invited to present their experience with specific tools and toolkits and discuss the results.

CONCLUSION

All stakeholders need to collaborate to develop innovative and practical solutions. There are many unanswered questions that necessitate further research but while there are gaps in knowledge which need to be filled, the clock is ticking for hospitals to implement changes to reduce COPD related hospital readmissions. We must proceed to share and implement best practices while striving to measure results and

Pulmonary Rehabilitation: Advances in the Past 10 Years

Richard Casaburi, PhD, MD
Rehabilitation Clinical Trials Center
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Torrance, CA

It has been the best of times. It has been the worst of times. Pulmonary rehabilitation has undergone a series of tectonic shifts in the past 10 years, each one improving the scientific basis of the therapy. What has emerged is an intervention unmatched in its potential to benefit COPD patients. At the same time, events in the United States have conspired to make pulmonary rehabilitation even less available to the people who need it. This essay will provide an overview of these developments and suggest how the field is likely to move forward.

In the past 10 years, pulmonary rehabilitation professionals have found their voice. Working through national and international organizations such as the American Association of Cardiovascular and Pulmonary Rehabilitation, the American College of Chest Physicians, the European Respiratory Society and the American Thoracic Society, high profile Statements, State-of-the-Art Reviews and Guidelines have been published[1-4]. Within the American Thoracic Society, pulmonary rehabilitation at long last achieved Assembly status, cementing its role as mainline therapy.

To distill the accumulated evidence, pulmonary rehabilitation has been documented to be the most effective way to decrease dyspnea, improve exercise tolerance and improve health-related quality of life in COPD. The magnitude of benefit in these outcomes well exceeds those for all available pharmacologic treatment options. Moreover, pulmonary rehabilitation has been shown to decrease exacerbations, hospitalization and other measures of health care use, reduce depression and anxiety and improve cognitive function and self-efficacy. Whether survival is improved is unclear, largely because randomized trials large enough and of sufficient duration to evaluate this outcome have yet to be performed.

Target: Dysfunction of the Limb Muscles

Although physical inactivity is of primary importance in limb muscle dysfunction development in COPD, other mechanisms such as inflammation, oxidative stress, nutritional deficiency and hypoxemia likely play a role[5].

Limb muscle dysfunction is prevalent in COPD and has important clinical implications such as reduced exercise tolerance; it is also strongly correlated with poor quality of life and reduced survival. There have been recent advances in our understanding of the extent and nature of the structural and biochemical alterations in limb muscles in patients with COPD and also in the mechanisms underlying these alterations.

Structural alterations include generalized fiber atrophy, a shift from type I (aerobic) fibers to type IIx (anaerobic) fibers, a decrease in capillarity, reduced mitochondrial density and functionality and increased apoptosis. Bioenergetic alterations include decreased aerobic capacity (but preserved glycolytic capacity). Both reactive oxygen and nitrogen species (ROS and RNS) are synthesized at higher rates, which may overwhelm tissue antioxidant capacity and yield oxidative stress. Oxidative damage may damage cell components, eventually leading to cell death; contractile performance may be thereby be impaired.

Muscle fatigue has been found to occur at low work rates in COPD. In a substantial fraction of COPD patients, even in those with severe disease, fatigue of the muscles of ambulation has been found to be the primary factor limiting exercise endurance. For these patients, improving lung function (e.g., by bronchodilator administration) would not be expected to increase exercise tolerance[6].

Of importance in clinical research, muscle fatigue is more likely to be a limiting factor in cycling exercise than in walking tasks. It deserves mention that muscle dysfunction worsens during exacerbations of COPD and recovery is slow.

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Limb muscle dysfunction in COPD is remediable, at least in part, by programs of exercise training. This remediation constitutes the primary physiologic target of pulmonary rehabilitation.

Target: Dynamic Hyperinflation

Exercise intolerance (and its consequent dyspnea) is often the chief complaint of COPD patients. Pulmonary rehabilitation successfully improves exercise tolerance; the physiologic mechanisms by which it does so have become clearer. Because flow rates during exhalation are slow, when exercise demands greater respiratory rate and tidal volume, a point is reached at which the time allotted for expiration is insufficient to complete the exhalation. The consequence is progressively increasing end-expiratory lung volumes, a phenomenon known as dynamic hyperinflation[7]. The problem comes when dynamic hyperinflation engenders end-*inspiratory* lung volumes that approach the maximum ability to inspire (the total lung capacity). Dyspnea increases markedly at this point, and exercise must terminate.

This understanding has led to the rational design of strategies to lessen dynamic hyperinflation as a way to improve exercise tolerance. It can be reasoned that any intervention that either allows a longer time for exhalation or speeds the rate of expiration will lessen the tendency for dynamic hyperinflation and, consequently, improve exercise tolerance[8]. Practical interventions have been found to accomplish dynamic hyperinflation reduction. Both bronchodilators and heliox breathing allow faster expiratory flow; supplemental oxygen slows breathing. Importantly, a program of pulmonary rehabilitation has also been found to reduce dynamic hyperinflation by means of slowing breathing[9]. The mechanism is thought to relate to reduction in lactic acidosis (a stimulus to breathing) at a given level of exercise, though other mechanisms may also be involved.

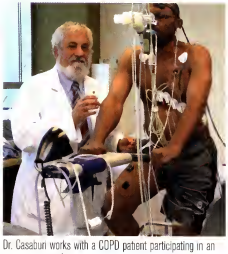
This line of evidence explains at least part of the mechanism by which pulmonary rehabilitation improves exercise tolerance. Importantly, it also suggests strategies that will allow rehabilitative exercise programs to be more effective. Because interventions reducing dynamic hyperinflation have been shown to be additive, performing exercise programs with the addition of optimal bronchodilation, heliox breathing or supplemental oxygen, should allow the COPD patient to tolerate higher exercise intensities and, thereby, induce greater gains in ambulatory muscle function. An interesting new option of breathing retaining through computerized ventilation-feedback displays[10], aimed at encouraging a slower deeper breathing pattern during exercise, may similarly reduce dynamic hyperinflation.

Exercise Training Strategies

Exercise training is the core of pulmonary rehabilitation. Training strategies have advanced as our understanding of the importance (and the mechanisms) of muscle dysfunction has evolved[5]. It is important to address deficits in both muscle endurance and strength as both are important in activities of daily living, though muscle endurance seems to be a more important determinant of overall function and quality of life. In general, endurance training (cycling, walking) increases endurance capabilities yielding increased aerobic enzyme concentrations, mitochondrial number and capillarity. Resistance training (weight lifting, calisthenics) increases muscle strength, yielding fiber hypertrophy. While the molecular mechanisms mediating the cause and effect relationship between muscular exercise and structural and biochemical changes remains mysterious, changes in certain mediators, such as insulin-like growth factor-1 (IGF1) and myostatin, seem likely to be involved[5].

Responses to exercise training among COPD patients seem quite variable; it is likely that there is a genetic component of this variability[11]. As to endurance training, a strong determinant of training responses is the tolerated exercise intensity. In addition to the strategies designed to decrease dynamic hyperinflation mentioned above, other approaches have been tried in an attempt to allow higher training intensities. Interval training, in which high intensity and low intensity exercise periods are alternated, allows (transient) toleration of work rates that could not be tolerated in a constant work rate task. Interestingly, in several studies, interval training was not found to be more effective

than constant work rate training[12], though studies continue. An interesting approach that seeks to allow increased intensity of training is to exercise one leg at a time. Some encouraging results have been obtained[13], but in the implementations employed to date, this seems an awkward way to exercise. Non-invasive ventilatory support has been shown to improve exercise tolerance[14] and may have the potential to enhance exercise training results.



Dr. Casaburi works with a COPD patient participating in an exercise research project.

Several other approaches (and adjuncts) to exercise training have begun to be explored:

- Resistance training is now routinely included in most pulmonary rehabilitation programs and seems to convey distinct functional benefits. The optimal balance between endurance and resistance components in a rehabilitative exercise session has yet to be defined.
- Rehabilitation early after a hospital admission for COPD exacerbation has started to be studied as a strategy to decrease the otherwise inevitable decline in muscle function and also as a possible method to prevent readmission[15].
- Neuromuscular electrical stimulation induces involuntary muscle contractions and has been shown to result in beneficial effects on muscle characteristics and function when applied over a time period similar to that used in an exercise training program[16]. This may be an attractive option for the most debilitated patient, perhaps serving as a bridge to a traditional exercise training program.
- Anabolic agents, mainly those that induce fiber hypertrophy, have had short-term trials in COPD[5]. Testosterone[17] and growth hormone has been studied. Novel androgen receptor agonists, agents that stimulate pituitary secretion of growth hormone and antibodies that inhibit the effects of myostatin are in early stages of evaluation. These drugs may eventually emerge as adjuncts to rehabilitative exercise programs.

Activity Promotion as an Independent Goal

It is unsurprising that COPD patients with severe disease live a sedentary existence. Avoiding the shortness of breath that activity brings is an understandable behavioral adaptation. However, studies have shown that even those with mild disease, in whom lung function would not be suspected to be limiting, have low activity levels[18]. Importantly, recent evidence indicates that, among COPD patients, the accustomed level of activity in everyday life is the strongest independent predictor of survival[19].

Pulmonary rehabilitation has always had a major focus on improving the ability to exercise and it has been supposed that this would translate into improved activity in everyday life. Testing this hypothesis has required development of methodology to assess activity levels. Activity questionnaires have been investigated, but patients generally over-report their level of activity. The past few years have seen a rapid development of activity monitoring technology. We now have unobtrusive monitors that can be worn for weeks at a time and fairly accurately reflect energy expended in daily activities; these monitors are enabling studies assessing whether available interventions are successful in increasing activity levels. Though the final word is not yet in, it appears that other therapies know to improve exercise tolerance in COPD (e.g., bronchodilators, supplemental oxygen) do not necessarily increase activity level[20,21]. This seems likely to reflect dissociation between the physiological ability to perform a task and actually performing the task in everyday life. Behavioral factors link these two and most COPD patients have had many years to adapt to a sedentary lifestyle; these habits are not easily broken.

Data on whether pulmonary rehabilitation increases physical activity levels is mixed. There are a dozen or so publications; roughly half showed significant increases in activity assessed by activity monitors following a rehabilitation program[20,22]. It seems likely that only a portion of patients undergoing pulmonary rehabilitation "get the message" and change their lifestyle. This may not be a totally disappointing – no therapy is effective in all people treated. However, it does point the way to modifications in the way

pulmonary rehabilitation is delivered. Behavior modification has always been an unspoken aim of rehabilitation programs. Is it time to draw on formal behavior modification strategies aimed specifically on increasing activity levels? Should the goal of exercise programs be switched from improving physiologic capabilities to instilling lifestyle changes? Should technology allowing monitoring of activity levels be utilized to accomplish these goals?

Maintaining the Benefits

Earlier demonstrations that the benefits of pulmonary rehabilitation tend to wane with time after the program was completed were greeted with some dismay. The expectation that rehabilitation would instill permanent benefits in all participants were likely unrealistic...consider that few (no?) therapies for chronic diseases yield benefits that last much beyond the period of administration. Recently, it has become apparent that the slow decline in benefits (over a year or two) among those who benefit from rehabilitation seen when group averages are considered may, in fact, represent two subgroups: in one benefits are largely sustained and in the other benefits quickly regress[53]. Identification of predictors of into which of the two subgroups a given patient is likely to belong would be helpful.

Maintenance programs, either conducted at home or as reduced-frequency supervised sessions, have been studied as methods to prolong the benefit of pulmonary rehabilitation. Though some encouraging data has been obtained[54], it is fair to say that the optimal format and timing of maintenance programs has not yet been established.

The Struggle for Wider Availability

Identifying strategies for paying for pulmonary rehabilitation has always been difficult. Perhaps because rehabilitation lacks the lobbying clout of, for example, the pharmaceutical industry, translating documentation of effectiveness into the willingness of payers to step forward to fund the therapy has been elusive. For many years, most programs in the United States were funded by a crazy quilt of strategies, depending on regional coverage policies. In some regions, it was virtually impossible to fund programs and, in these areas, pulmonary rehabilitation programs were rare. In 2010, a sustained campaign led by pulmonary rehabilitation professionals, led the Center for Medicare Services (CMS) to establish a national policy for pulmonary rehabilitation coverage. The rejoicing in the pulmonary rehabilitation community was short-lived, however. After not much longer than a year, an accounting of costs incurred by hospitals for pulmonary rehabilitation therapy reached CMS. This accounting was interpreted as indicating that CMS payments were overgenerous; payments were promptly slashed to a point where many programs became non-viable. Pulmonary rehabilitation leadership promptly pointed out the flaws in the data CMS had relied on, but we anticipate a long wait before the situation is remedied.

A problem attendant to an underfunded therapy is that practitioners are not attracted to the field. It is hard to see where the next generation of pulmonary rehabilitation program leaders will come from. There is no defined educational program in the United States to prepare individuals for a career in pulmonary rehabilitation. This seems to be less true in Europe, where Physiotherapy seems to offer a pathway for pulmonary rehabilitation professionals.

Interestingly, pulmonary rehabilitation is not particularly expensive; the cost of a full program is roughly comparable to the cost of a year's supply of a maintenance bronchodilator. Pulmonary rehabilitation is most commonly delivered in an outpatient facility in (or adjacent to) a hospital. Recent efforts have focused on finding less expensive alternatives. Having patients undergo the majority of their rehabilitation program at home has the attraction of a lower cost, but also some potential deficits. Without the ongoing motivational support of rehabilitation professionals and without surrounding the patient with similarly afflicted individuals, it is hard to see how home programs can be equally effective. It seems possible that technologic innovation (e.g., videoconferencing, step counters, activity diaries) might make home rehabilitation an option in areas where no in-center program is available.

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New Open Access, Online COPD Journal Coming this May

Cathy Carlomagno
Production Editor, Chronic Obstructive Pulmonary Diseases: Journal of the COPD Foundation

As announced in the fall, the COPD Foundation will launch a new "open access," online journal devoted to COPD this spring.

Offering the lung health community an "open access" premier COPD journal is a long awaited vision that will become a reality for the COPD community when the first issue of the *Chronic Obstructive Pulmonary Diseases: Journal of the COPD Foundation* (JCOPDF) is posted online in May. The launch of this inaugural issue coincides with the Foundation's 10th Anniversary.

"I am confident that this new journal will create an international dialogue that will foster improved diagnosis and care for patients with all forms of chronic obstructive pulmonary diseases," says James Crapo, M.D., Professor of Medicine at National Jewish Health and the University of Colorado, Denver, a principal investigator of the COPDGene® Study, and the Journal's Editor-in-Chief. "This new publication is not only an exciting new venture for the Foundation, but the perfect way to commence the Foundation's next 10 years."

The Journal will publish original research articles, basic and clinical review articles, perspectives/short communications, practice guidelines related to COPD and letters to the editor.

Submitted articles will undergo rigorous peer review under the direction of Dr. Crapo, the Journal's other editors—Peter J. Barnes, D.M., D.Sc., (National Heart and Lung Institute--UK), Paul Jones, M.D., Ph.D., (St George's University of London), David M. Mannino, M.D., (University of Kentucky) and Barry J. Make, M.D., (National Jewish Health)—and its international editorial board. (To view a list of this editorial board, visit: www.copdfoundation.org/Learn-More/For-Medical-Professionals/Journal.aspx)

"Our goal as we convened this editorial board was to include thought leaders worldwide who have shaped the field of COPD and will continue to over the coming decade," explains Crapo.

After launch of the inaugural May issue, subsequent issues will be published online quarterly however, individual articles will be published rapidly online following peer review and editorial acceptance.

"The COPD Journal provides the COPD community with an outlet for high-quality articles that can be accessed quickly and easily," says Crapo. "It's vital that information about COPD research and medical discoveries be shared with a wider audience, from clinical trials to observational epidemiologic and genetic studies, because shared knowledge serves as the catalyst for medical breakthroughs."

Some of the topics to be addressed via original research and clinical review articles in the first issue are:

- new CT findings from the COPDGene study;
- a dietary intake association with COPD phenotypical characteristics;
- a pilot study on short-term, high intensity exercise;
- co-morbidities and racial disparities;
- and MORE....

"Our goal with this Journal is to be topical and relevant to not only pulmonologists but also internists, primary care specialists, basic and clinical researchers, and all health care professionals with an interest in chronic obstructive pulmonary diseases," Crapo says.

To register to receive a free subscription to the Journal and regular updates, visit http://www.copdfoundation.org/Learn-More/For-Medical-Professionals/Journal.aspx. To offer suggestions for Dr. Crapo and the editorial board, send an email to copd@njhealth.org.

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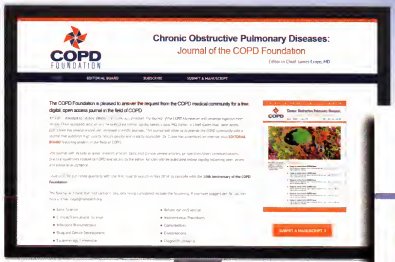
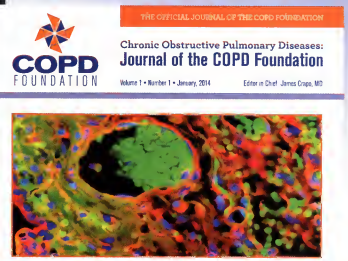
COPD Foundation Launches Open Access Scientific Journal





Chronic Obstructive Pulmonary Diseases: Journal of the COPD Foundation (JCOPDF) is a free, digital and open access journal. JCOPDF will publish quarterly, with the first issue launching in May 2014 to coincide with the Foundation's 10th Anniversary.

James Crapo, M.D., editor-in-chief, COPD Journal, has assembled an international editorial board featuring leaders in the COPD field. Articles submitted to the COPD Journal will undergo rigorous peer review, and once accepted will be published online.

Visit the journal website to register to be notified when new articles are posted, or to submit a manuscript.

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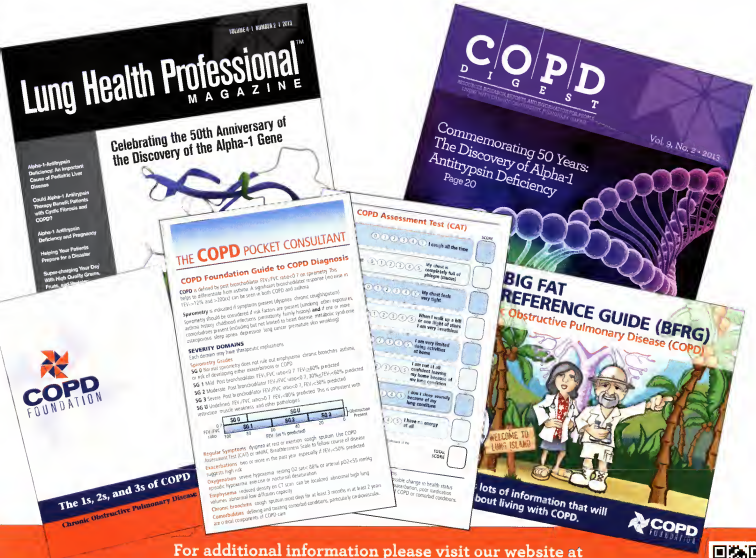

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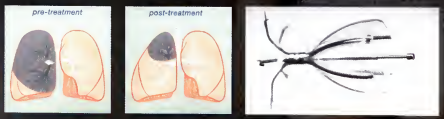

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Spiration, Inc., of Redmond, Washington, is the sponsor of the EMPROVE Clinical Trial.

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IN ALPHA-1 ANTITRYPSIN AUGMENTATION
THE FIRST AND ONLY

READY TO INFUSE

LIQUID GLASSIA[1-4]

FEWER STEPS[1]

NO RECONSTITUTION

SAFETY CONSIDERATIONS[1,5]

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One ASHP recommendation to reduce preparation errors is to "dispense medications in ready-to-administer dosage forms whenever possible."[5]

The American Society of Health-System Pharmacists (ASHP)
Guidelines on Preventing Medication Errors in Hospitals

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INDICATION FOR GLASSIA [ALPHA$_1$-PROTEINASE INHIBITOR (HUMAN)]

GLASSIA is indicated for chronic augmentation and maintenance therapy in individuals with emphysema due to congenital deficiency of alpha$_1$-proteinase inhibitor (Alpha$_1$-PI), also known as alpha1-antitrypsin (AAT) deficiency.

- The effect of augmentation therapy with GLASSIA or any Alpha$_1$-PI product on pulmonary exacerbations and on the progression of emphysema in Alpha$_1$-PI deficiency has not been demonstrated in randomized, controlled clinical trials.
- Clinical data demonstrating the long-term effects of chronic augmentation and maintenance therapy of individuals with GLASSIA are not available.
- GLASSIA is not indicated as therapy for lung disease in patients in whom severe Alpha$_1$-PI deficiency has not been established.

DETAILED IMPORTANT RISK INFORMATION FOR GLASSIA

GLASSIA is contraindicated in immunoglobulin A (IgA) deficient patients with antibodies against IgA. Patients with selective or severe IgA deficiency and with known antibodies to IgA have a greater risk of developing severe hypersensitivity and anaphylactic reactions.

GLASSIA is contraindicated in individuals with a history of severe immediate hypersensitivity reactions, including anaphylaxis, to Alpha$_1$-PI products.

Monitor vital signs continuously and observe the patient carefully throughout the infusion. **If anaphylactic or severe anaphylactoid reactions occur, discontinue the infusion immediately.**

GLASSIA is made from human plasma and may carry a risk of transmitting infectious agents, such as viruses, and theoretically, the Creutzfeldt-Jakob disease (CJD) agent.

GLASSIA should be administered at room temperature at a rate not greater than 0.04 mL/kg body weight per minute. Administer GLASSIA within 3 hours of entering the vials.

Safety and effectiveness in patients over 65 years of age have not been established.

In the clinical studies, one subject experienced a treatment emergent serious adverse reaction (infective exacerbation of COPD), considered possibly related to treatment with GLASSIA due to its temporal association. The most common adverse reactions deemed possibly related to GLASSIA administration (>5%) were headache and dizziness.

Please see Brief Summary of Full Prescribing Information on the adjacent page.

References: 1. GLASSIA [Alpha$_1$-Proteinase Inhibitor (Human)] Prescribing Information. Westlake Village, CA: Baxter Healthcare Corporation; June 2012. **2.** ZEMAIRA [Alpha$_1$-Proteinase Inhibitor (Human)] Prescribing Information. CSL Behring, LLC: Kankakee, IL; April 2013. **3.** ARALAST NP [Alpha$_1$-Proteinase Inhibitor (Human)] Prescribing Information. Baxter Healthcare Corporation: Westlake Village, CA; April 2010. **4.** PROLASTIN-C [Alpha$_1$-Proteinase Inhibitor (Human)] Prescribing Information. Talecris Biotherapeutics, Inc. Research Triangle Park, NC; January 2013. **5.** ASHP guidelines on preventing medication errors in hospitals. American Society of Health System Pharmacists Web site. http://www.ashp.org/s_ashp/docs/files/MedMis_Gdl_Hosp.pdf. Accessed June 18, 2013.

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February 2014 USBS/341/13-0003a

Glassia
[Alpha$_1$-Proteinase Inhibitor (Human)]

Baxter

GLASSIA [Alpha$_1$-Proteinase Inhibitor (Human)]

Brief Summary of Prescribing Information. Please see package insert for full prescribing information.

INDICATIONS AND USAGE

Alpha$_1$-Proteinase Inhibitor (Human), GLASSIA is indicated for chronic augmentation and maintenance therapy in adults with emphysema due to congenital deficiency of alpha$_1$-proteinase inhibitor (Alpha$_1$-PI), also known as alpha$_1$-antitrypsin (AAT) deficiency.

- The effect of augmentation therapy with GLASSIA or any Alpha$_1$-PI product on pulmonary exacerbations and on the progression of emphysema in Alpha$_1$-PI deficiency has not been demonstrated in randomized, controlled clinical trials.
- Clinical data demonstrating the long-term effects of chronic augmentation and maintenance therapy of individuals with GLASSIA are not available.
- GLASSIA is not indicated as therapy for lung disease in patients in whom severe Alpha$_1$-PI deficiency has not been established.

DOSAGE AND ADMINISTRATION

- **For Intravenous Use Only.**
- Use aseptic technique for all preparation and administration steps.
- Inspect the vial of GLASSIA. The solution should be clear and colorless to yellow-green and may contain a few protein particles. Do not use if the product is cloudy.
- Administer GLASSIA alone; do not mix with other agents or diluting solutions.
- Administer product brought to room temperature within three hours of entering the vials.

Treatment of Congenital Alpha$_1$-Proteinase Inhibitor Deficiency

The recommended dosage of GLASSIA is 60 mg/kg body weight administered once weekly by intravenous infusion. Dose ranging studies using efficacy endpoints have not been performed. The recommended dosage of 60 mg/kg takes approximately 60-80 minutes to infuse. The infusion rate should not exceed 0.04 mL/kg body weight per minute.

CONTRAINDICATIONS

GLASSIA is contraindicated in immunoglobulin A (IgA) deficient patients with antibodies against IgA.

GLASSIA is contraindicated in individuals with a history of severe immediate hypersensitivity reactions, including anaphylaxis, to Alpha$_1$-PI products.

WARNINGS AND PRECAUTIONS

Hypersensitivity to IgA

GLASSIA may contain trace amounts of IgA. Patients with selective or severe IgA deficiency and with known antibodies to IgA, have a greater risk of developing severe hypersensitivity and anaphylactic reactions. Monitor vital signs continuously and observe the patient carefully throughout the infusion. **IF ANAPHYLACTIC OR SEVERE ANAPHYLACTOID REACTIONS OCCUR, DISCONTINUE THE INFUSION IMMEDIATELY.** Have epinephrine and other appropriate supportive therapy available for the treatment of any acute anaphylactic or anaphylactoid reaction.

Transmissible Infectious Agents

Because this product is made from human plasma, it may carry a risk of transmitting infectious agents, such as viruses, and theoretically, the Creutzfeldt-Jakob disease (CJD) agent. The risk of transmitting an infectious agent has been minimized by screening plasma donors for prior exposure to certain viruses, by testing for the presence of certain current virus infections and by inactivating and removing certain viruses during the manufacturing process (see *Description [11]* in full prescribing information for viral reduction measures). Despite these measures, such products may still potentially transmit human pathogenic agents. There is also the possibility that unknown infectious agents may be present in such products.

The physician should weigh the risks and benefits of the use of this product and discuss the risks and benefits with the patient.

All infections thought by a physician possibly to have been transmitted by this product should be reported by the physician or other healthcare provider to Kamada Ltd. at 1-866-GLASSIA or FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.

No seroconversions for hepatitis B or C (HBV or HCV) or human immunodeficiency virus (HIV) or any other known infectious agent were reported with the use of GLASSIA during the clinical studies.

ADVERSE REACTIONS

The serious adverse reaction observed during clinical studies with GLASSIA was exacerbation of chronic obstructive pulmonary disease (COPD).

The most common drug-related adverse reactions considered by the investigator to be at least possibly related to GLASSIA administration observed at a rate of >5% in subjects receiving GLASSIA were headache and dizziness.

Adverse Reactions[1] Occurring in > 5% of Subjects During the First 12 Weeks of Treatment

	GLASSIA No. of subjects: 33	Prolastin No. of subjects: 17
Adverse Event (AE)	**No. of subjects with adverse reactions[1] (AR) (percentage of all subjects)**	**No. of subjects with adverse reactions[1] (AR) (percentage of all subjects)**
Cough	3 (9%)	4 (24%)
Upper respiratory tract infection	3 (9%)	0 (0%)
Headache	3 (9%)	3 (18%)
Sinusitis	2 (6%)	1 (6%)
Chest discomfort	2 (6%)	0 (0%)
Dizziness	2 (6%)	0 (0%)
Hepatic enzyme increased	2 (6%)	0 (0%)

[1]An adverse reaction is any adverse event which met any of the following criteria (a) an adverse event that began within 72 hours following the end of product infusion, or (b) an adverse event considered by either the investigator or sponsor to be at least possibly related to product administration, or (c) an adverse event for which causality assessment was missing or indeterminate.

Postmarketing Experience

The following reactions have been identified during postmarketing use of GLASSIA in clinical practice. Because they are reported voluntarily from a population of unknown size, estimates of frequency cannot be made. The reactions, which have been chosen for inclusion due to either their seriousness, frequency of reporting, possible causal connection to GLASSIA, or a combination of these factors, include: Headache, Dyspnea, Fatigue and Nausea.

USE IN SPECIFIC POPULATIONS

Pregnancy

Pregnancy Category C

Animal reproduction studies have not been conducted with GLASSIA. It is also not known whether GLASSIA can cause fetal harm when administered to pregnant women or can affect reproductive capacity. GLASSIA should be given to a pregnant woman only if clearly needed.

Nursing Mothers

It is not known whether Alpha$_1$-PI is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when GLASSIA is administered to a nursing woman.

Pediatric Use

Safety and effectiveness in pediatric patients have not been established.

Geriatric Use

Clinical studies of GLASSIA included 11 subjects of 65 years of age or older. This number of subjects was not sufficient to determine whether they respond differently from younger subjects. As for all patients, dosing for geriatric patients should be appropriate to their overall situation. Safety and effectiveness in patients over 65 years of age have not been established.

PATIENT COUNSELING INFORMATION

- Inform patients of the early signs of hypersensitivity reactions, including hives, generalized urticaria, chest tightness, dyspnea, wheezing, faintness, hypotension, and anaphylaxis. Advise patients to discontinue use of the product and contact their physician and/or seek immediate emergency care, depending on the severity of the reaction, if these symptoms occur.
- Inform patients that GLASSIA is made from human plasma and may contain infectious agents that can cause disease (e.g., viruses and, theoretically, the CJD agent). Explain that the risk of GLASSIA transmitting an infectious agent has been reduced by screening the plasma donors, by testing the donated plasma for certain virus infections, and by a process demonstrated to inactivate and/or remove certain viruses during manufacturing (see *Warnings and Precautions*). Symptoms of a possible virus infection include headache, fever, nausea, vomiting, weakness, malaise, diarrhea, or, in the case of hepatitis, jaundice.
- Inform patients that administration of GLASSIA has been demonstrated to raise the plasma level of Alpha$_1$-PI, but that the effect of this augmentation on the frequency of pulmonary exacerbations and on the rate of progression of emphysema has not been established by clinical trials.

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Progress in Lung Cancer Screening over the past 10 years

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Associate Professor of Medicine
Baylor College of Medicine
Staff Physician, Michael E. DeBakey VA Medical Center
Houston, Texas

Introduction

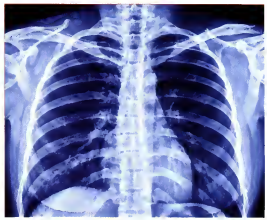
Lung cancer is the second most common type of cancer and the leading cause of cancer death in adults. Mortality due to lung cancer has grown over years. The American Cancer Society estimates about 228,190 new cases of lung cancer and 159,480 deaths in 2013 in USA (1). Worldwide, lung cancer kills about 1.38 million individual (18.2% of cancer death)(2). Tobacco smoking is the major risk factor leading to lung cancer and an important risk factor for cancer in general. About 81% of lung cancers are in former or current smokers while the rest is in individuals who never smoked. Other risk factors for lung cancer include exposure to second-hand cigarette smoker, occupational exposure to asbestos, nickel, chromium or arsenic, and radiation exposure (1;3). Furthermore, the presence of chronic obstructive pulmonary disease (COPD) increases the risk of lung cancer.

Lung cancer survival is the lowest among the four prevalent cancers including breast, prostate and colon cancers. Furthermore, the five-year survival rate has not changed appreciably in last several decades (13% in period 1974-1976 vs. 15.9% in 2002-2008). One reason for this grave outcome is late diagnosis of lung cancer. Up to 56% of patient with lung cancer at the time of diagnosis has metastasis compared to only 4% in prostate, 5% in breast, and 20% in colon cancer. Five-year survival rate is as high as 60-75% in individuals diagnosed at earlier stages (Stage I disease) [4]. With this in mind, approaches that result in earlier diagnosis of lung cancer will be significant step toward improved outcome in treatment of lung cancer. However, an effective screening intervention should reduce the mortality without causing harm with reasonable cost [5]. A recent trial of low dose chest CT scan demonstrated promise. In this paper we review the advances made during last decade in screening of lung cancer and explore the current recommendations for lung cancer screening.

New Data about lung cancer screening in last decade

1) Chest X- Ray (CXR)

In clinical practice, investigation for lung cancer usually starts with symptomatic patients and thus, in a



very large number of patients the disease is already advanced. CXR has been used in both research and clinical setting to screen for lung cancer. Prior randomized trials evaluated role of CXR on lung cancer mortality. CXR on regular intervals resulted in more lung cancer detection but there was no appreciable difference in cumulative lung cancer mortality [6]. A recent advance in the last decade was publication of a large NIH sponsored study titled "The Prostate, Lung, Colorectal, and Ovarian (PLCO) Randomized Trial". This study randomized 154,901 subjects to annual screening with CXR versus usual care. The screening included CXR for 4 years. The study showed that annual screening with CXR did not decrease lung cancer mortality when compared to usual care [7]. A recent systematic review of literature of controlled studies did not

This article is special for the COPD Foundation's 10-year anniversary.

find any support for CXR as a screening tool for lung cancer detection [5].

2) Sputum cytology

Along CXR several studies evaluated role of sputum cytology in screening of lung cancer. The U.S. preventive Services Task force extensive review of existing literature did not find any support for sputum cytology alone or added to CXR in screening of lung cancer [5,8]. A recent extensive review by American College of Chest Physicians did not recommend sputum cytology [6]. However, newer technical developments in assessing various markers in the sputum may provide better screening tools in future. The newer technologies include abnormal DNA methylation, abnormal immune-staining patterns, monoclonal antibodies and identification of genetic mutations may help to identify individuals with early lung cancer [8,9]. Role of the new sputum biomarkers is not known at this time.

3) Biomarkers of lung cancer

Recent scientific advances in lung cancer diagnosis and treatment identified various diagnostic biomarkers that may play a role in early diagnosis of lung cancer. These biomarkers may be measured in exhaled breath, in sputum and in blood or urine. Although data from preliminary studies look promising, value of these biomarkers in screening lung cancer has not been studied and is in need of more clarification [7,10]. Investigators from National Lung Screening Trial (NSLT) and the American College of Radiology Imaging Network (ACRIN) collected various biospecimen repository including samples from blood, sputum and urine from more than 10,000 of participants of the low dose CT scan trial. This repository may help to assess role of newly discovered biomarkers in early diagnosis of lung cancer [9].

4) Low Dose Chest CT Scan

With the advent of imaging technology, chest CT scan has become a common diagnostic tool in diagnosis and management of various cancers including lung cancer. Many studies evaluated role of Chest CT in diagnosis of early lung cancer. The most important news in screening of lung cancer in the past decade is more clarification on role of low dose chest CT (LDCT) in diagnosis of lung cancer in asymptomatic high risk individuals.

A large study funded by National Institute of Health and conducted by major academic institutes evaluated role of LDCT in a very specific at risk population. National Lung Screening Trial (NSLT) randomized 53,454 individuals to LDCT or single view CXR. Inclusion criteria included age between 55-74 (at the time of randomization), history of at least 30 pack year smoking, current smoker or past smokers who quit within the previous 15 years[10]. The study participants underwent yearly imaging for three years. The study showed a significant 20% reduction in lung cancer mortality in the LDCT group compared to CXR group (Relative Risk of 0.80; with 95% confidence interval of 0.73-0.93). The significant finding resulted in new recommendations by various societies. The U.S. preventive task force in its 2004 statement did not find sufficient data indicating any mortality benefit from LDCT in screening of lung cancer [8]. However, recent updated report from the task force recommended LDCT as a screening method in asymptomatic adults aged 55 to 80 who meet the criteria similar to NSLT (Ann Intern Med, Moyer, 2013) [11]. Similarly, American College of Chest Physicians in an updated statement recommended LDCT as a screening tool in individuals who meet the inclusion criteria used in NSLT [6]. Interestingly and rightfully, the statement by ACCP emphasizes the importance of practice setting and the expertise available in the screening centers in its recommendation.

Although NSLT clarified rule of LDCT in the study population, rule of LDCT in other populations including younger individuals or those with less exposure is not clear yet. Prior smaller studies that enrolled different study participants did not show mortality benefit [12,13] . Thus, caution should be exercised in expanding results of LDCT to individuals different that those enrolled in NSLT. Fortunately, results from other ongoing trials may help to define other populations that may benefit from LDCT [6].

The NSLT clearly showed that the LDCT in the specific population and in the specific

study setting provides mortality benefit. However, rush to a widespread use of the data may not result in similar outcome. As rightfully mentioned by Detterbeck and colleagues, there are many factors that should be considered before widespread implementations of LDCT [1]. Various expert societies involved in diagnosis and management of lung cancer have enlisted various elements for a successful LDCT screening program. Several factors are common to this recommendations including multispecialty nature of the program, development and establishment of patient screening processes, quality control of LDCT, establishing uniformity in interpretation of the scans, and developing intervention algorithm. One important recommendation from ACCP statement is "demonstration projects" in each specific practice setting to ease implementation and trouble shooting of the LDCT screening program [1].

In summary, various clinical trials and cohort studies in the last decade or so clarified role of various clinically available tools in screening of asymptomatic patients at high risk of lung cancer. Recent data does not provide any support for CXR and sputum cytology in screening of lung cancer. However, use of biomarkers in various specimens, including sputum samples, may prove to be helpful in future. Result of NSLT clearly showed mortality benefits for screening of lung cancer in a specific at risk population. The study was conducted in large academic medical center with access to advanced care. Extending the results of NSLT to other health care environment should be done with caution. Particularly, use of demonstration projects will help to implement the screening program more effectively and increases the likelihood of similar outcomes to NSLT.

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COPD8USA-Online CME Opportunity

The COPD8USA Scientific Planning Committee Members and the COPD Foundation are pleased to provide you with an opportunity to receive continuing medical education credits. This online activity is based on content from the COPD8USA conference, held in June 2013 in Chicago, Illinois.

COPD8USA was the second in a series of biennial conferences modeled after the successful COPD Conference Series held in Europe for the past 16 years. Founded by international experts, Professors Robert Stockley and Sue Hill, the COPD Conference Series has helped thousands of clinicians and researchers improve the care of their patients living with COPD worldwide. Unlike other COPD conferences that focus solely on the scientific and research aspects of COPD, the COPD Conference Series is designed to provide practical information for clinical management as well as the latest in clinical and basic research.

The COPD Foundation is committed to improving the quality of life for individuals affected by COPD. COPD8USA reflected this commitment through interactive, practical and interprofessional educational opportunities. We realize that not everyone was able to attend the full conference, so we are pleased to be able to offer learners the opportunity to participate in this online activity. We hope that this activity will provide you with a snapshot of the exciting learning opportunities that were offered at the full conference.

The following sessions from COPD8USA will be featured in the online CME activity:

- **Detecting and Treating COPD Comorbidities: A Path to Improving Overall Health**
- **The Future of COPD in America**
- **2013 COPD Guidelines – Where Are We Headed?**
- **Management of Advanced COPD – A Panel Discussion**
- **Nature Versus Nurture in COPD**
- **Moving COPD Phenotypes into Clinical Practice**
- **COPD Hospital Readmissions: Understanding the Causes, Identifying the Solutions, and Measuring the Results**

The COPD Foundation is honored to serve as the host for the only COPD conference of this kind. We plan to host the COPDUSA conference every other year (odd calendar years). We are grateful to the world class faculty and all of the attendees who supported the conference. The need for focused COPD education for the entire healthcare provider team has never been greater.

We are looking forward to hosting COPD9USA in Chicago on June 5-6, 2015. If you are interested in receiving information about the COPD9USA conference, please register at www.copdconferencesusa.org and you will be notified when the conference registration for COPD9USA is live. For more information, please contact Elisha Malanga: emalanga@copdfoundation.org.

The COPD Foundation extends a special thanks to Robert Stockley, M.D., Professor Sue Hill, and the COPD Conference Committee from the UK for their partnership and support.

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[1] Zuberhuhler P, et al. In vitro testing of new non-electrostatic holding chamber with hydrofluoralkane salbutamol and beclomethasone inhalers. CHEST 2002; 122:1855

[2] A N. Weigand, L. Cambridge, N H. Tiffin, U. Schuschnig. Using RDDR to Compare Delivery Efficiency of Three Commercially Available Breath-Enhanced Nebulizers with Budesonide. American College of CHEST Physicians. October 2008

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JUNE 5–6, 2015

CHICAGO

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www.copdconferencesusa.org

Attendees of the COPD9USA conference will explore improving outcomes in the management of COPD through scientific presentations and unique networking opportunities.

WHO SHOULD ATTEND:

- Primary Care Physicians
- Clinical/Basic Scientists
- Respiratory Therapists
- Managed Care Leaders
- Pulmonologists
- Physical Therapists
- Physician Assistants
- Public Health Professionals
- Advanced Practice/ Registered Nurses

COPD FOUNDATION

University of Nebraska Medical Center

The COPD Foundation Celebrates a Decade of Progress

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Director of Education, COPD Foundation
Washington, DC
John W. Walsh
President and Co-Founder, COPD Foundation
Washington, DC

This year marks the 10th anniversary of the COPD Foundation, and a decade of progress that we've established, built and will continue to grow upon. This article features four different perspectives of the past decade of COPD from prominent individuals in the COPD community: COPD Foundation President and Co-Founder John W. Walsh, COPD Foundation Board Member Gerard Turino,M.D., COPD Foundation Director of Education Scott Cerretta, R.R.T, and Patricia A. Jellen, M.S.N, R.N

Patricia A. Jellen, M.S.N., R.N.:

Ten years seems like an eternity, but in reality, the time goes very fast and so much can happen! When asked to reflect on the past 10 years as it related to COPD and me, a registered nurse, I am filled with excitement and pride. While we still face obstacles in managing and treating COPD, I can honestly say that many positive things have happened over the past 10 years. Advances in research, advocacy and disease management have moved us in a positive direction in the COPD world.

In 2003, my primary role, as it related to COPD, was coordinating the lung volume reduction surgery (LVRS) program at New York Presbyterian Hospital-Columbia University Medical Center(NYPH/CUMC). A major part of my responsibilities between 1998 and 2003 included coordinating our institution's participation in the National Emphysema Treatment Trial (NETT). The NETT results, released in May 2003, were able to give guidance as to the role of LVRS in the treatment of emphysema. For the first time ever, we had objective information about which patients could potentially benefit from the procedure. Based on the NETT results, providers could categorize potential surgical candidates into groups to give better predictions of benefit and potential risk from LVRS. NETT defined those patients who were at increased risk of death and who should never be offered LVRS. The Centers for Medicare and Medicaid Services (CMS), utilizing NETT results, amended its coverage policy for LVRS and established clear, concise guidelines for those beneficiaries/patients for whom the procedure of LVRS could be considered and covered.

January 2004 marked the effective date for the initiation of the national coverage policy for Medicare beneficiaries for LVRS. Since 2004, the LVRS program at NYPH/CUMC continues to evaluate and operate on patients who meet the NETT criteria. Our outcomes mirror those from NETT and we continue to look for new ways to improve the quality of the lives of the COPD patients referred to our institution.

While the number of patients who get LVRS nationwide today remains relatively low, I would say that the validation of LVRS as a potential therapy or intervention for patients with COPD has generated innovative development and research of alternate methods to achieve the same results of LVRS but with a less invasive approach.

Such awareness of research comes from the huge growth I have seen in advocacy from the COPD community over the past 10 years. Finally, those affected with COPD have resources and groups that can help them navigate an often complicated health care system and improve the management of their health or specifically their COPD. The mere fact that the COPD Foundation is where it is today is a testament to the people, patients and providers, who have remained steadfast in their belief that they can make a difference. I credit advocacy and patient awareness over the past 10 years with changes in the development of oxygen devices to allow for increased mobility; improvement in the ability to travel with oxygen; and finally with the fact that today we have a national policy for the coverage of pulmonary rehabilitation. Increased

This article is special for the COPD Foundation's 10-year anniversary.

patient awareness and involvement is key.

As a nurse I know how important patient education is to outcomes, and my experience working with patients who have COPD over the past 16 years has taught me that knowledge related to medications, proper inhaler technique, signs and symptoms of an exacerbation, hand hygiene, exercise, oxygen therapy, and energy conservation is critical to outcomes in those with COPD. People must be empowered through knowledge to manage their own disease. The development and availability of educational resources for COPD has been amazing over the past decade. Such educational materials, websites and blogs allow patients and their caregivers to learn about COPD. Patients now are learning to LIVE with COPD, not merely exist with it.

So over the past decade, we have accomplished much but we are not done. The groundwork has been laid, now is it time that we push forward. We should not be satisfied with the status quo. We, the COPD community, must demand more. Let's go for it all- funding for the cure, stem cell research–whatever it takes to change the tide. Imagine what someone will be writing about in 2024.

Gerard Turino, M.D.:

I began my career as a pulmonary physician with a fellowship in the 1950s and was fortunate enough to have my early clinical training at the Columbia University Division at Bellevue Hospital in New York. At that time, Bellevue was a center of cardiopulmonary research in the United States and beyond. The directors of that center were Drs. Dickinson Richards and Andre Cournand who, with Werner Forsmann were the first to catheterize the human heart in patients. For this advance, they received the Nobel Prize in Medicine and Physiology in 1956.

At that time, one could characterize the clinical understanding of COPD as undefined. What understanding there was came mostly from anatomic pathology (a medial specialty that is concerned with the diagnosis of disease based on different examinations of organs and tissues), which described the presence of emphysema in lungs of patients dying with lung disease. Such a description was first recorded by the famous French pathologist Laennec in the early 1800's. Bronchi were recognized as being narrowed and inflamed and abnormalities in the structure of the elastic tissue were so noted in the early 1960's. In the 1940's and early 1950's pulmonary function tests were being developed with great precision. Lung function tests could quantify the obstruction to bronchial airflow, the lowering of the capacity for respiratory gas exchange and the changes in mechanical characteristics of the lung in terms of a loss of lung recoil. However, characterization of COPD as a clinical entity in patients varied among clinicians.

The following statement appeared in the introduction to a publication on diagnostic standards from the American Thoracic Society in 1962 entitled: *Chronic Bronchitis, Asthma and Pulmonary Emphysema.*

"Because precise definitions and classifications have not been available, a tendency has developed for workers in separate disciplines to define these diseases differently by criteria selected from their specific frames of reference or by their particular methods of observation or techniques for measurements. The lack of widely accepted definitions and classifications has impaired communication which, in turn, has retarded clinical and research progress."

At that time in England, with its cold and rainy climate, chronic bronchitis was thought to be the predominant cause of lung disease because of its manifestation of daily cough and sputum. In the Netherlands, there was emphasis on allergic abnormalities and the role of airway hyper-reactivity as related to asthma. In the U.S., there was recognition that anatomical emphysema was a significant element in the disease. Smoking, as a causative factor, was not to be accepted as a toxic agent until the early 1960's.

However, a major insight into disease mechanisms came in 1963 when alpha-1 antitrypsin deficiency was discovered by Laurell and Eriksson in Sweden as a genetic abnormality associated with the development of emphysema in men and women at an early age and in the absence of exposure to tobacco smoke. The genetically determined decreased levels of this protein in blood and tissues in such patients meant that a significant elastase, an enzyme present in neutrophils (white blood cells) which chemically degrade elastin, could not be inhibited adequately in the body. Elastin

in the lung, which is an essential structural element for alveoli, would be degraded and alveolar architecture disrupted.

A protease-antiprotease imbalance hypothesis took shape from that time. This same imbalance could be applied also to patients with COPD who had normal levels of alpha-1 antitrypsin in blood and tissue when it was shown that the increased oxidants in smokers could functionally inactivate the inhibitory capacity of alpha-1 antitrypsin protein.

To formalize the clinical approach to COPD, the American Thoracic Society convened a workshop on definitions of COPD in 1984. This workshop was chaired by Gordon Snider and I was also a member. Out of this workshop definitions were developed which still apply today. Emphysema was defined as an anatomical diagnosis in which there was destruction of alveolar structure, which had to be established by anatomical criteria or radiological criteria. Bronchitis was defined as the presence of cough and sputum on most days for three months each year for two years. Clinical characterization of COPD was the presence of irreversible airway obstruction with an FEV1 below 70% predicted. Along the way, a third diagnostic component was added to chronic bronchitis and emphysema under the umbrella of COPD, which is refractory asthma recognizing that some patients with persistent asthma go on to develop chronic airway obstruction.

Significant position papers on the diagnosis and treatment of COPD were published by the American Thoracic Society in 1995 and updated in 2004. Also, other highly significant position papers were published as "The Global Strategy for the Diagnosis, management and presentation of chronic obstructive pulmonary disease", first published in 2001 and revised in 2006. This GOLD Strategy, as it was called, formalized the clinical status of patients as determined by spirometric performance involving measurements of FEV1 and FEV1/FVC with gradations of mild, moderate, severe and very severe disease. The cut-off between normal and COPD is, by Gold criteria, FEV1/FVC < 70%. The GOLD criteria offered the following definition of COPD:

"Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease with some significant extrapulmonary effects that may contribute to the severity in individual patients. Its' pulmonary component is characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases."

The development of the "GOLD Criteria." was significant in providing a common language for clinicians to classify patients for clinical care and projects in research.

Beginning 10 years ago, there was the realization that COPD was becoming a major public health problem, which was going unrecognized by patients and by physicians since mild or moderate forms of airway obstruction were being unrecognized by patients and attributed to weight gain, age or deconditioning. There was also the realization that COPD should best be diagnosed early to eliminate tobacco exposure and treat exacerbations.

John Walsh, an alpha-1 antitrypsin deficiency patient himself and founder of the Alpha-1 Foundation recognized that a new foundation focusing on COPD specifically, as a major public health problem nationally and internationally, could serve medicine and the public good. The COPD foundation was thus formed in the year 2004 with the mission to improve patient care, increase public awareness, augment education with respect to COPD among physicians and medical personnel and promote research to increase understanding COPD to achieve a cure. From its beginning the COPD Foundation worked cooperatively with the American Thoracic Society, the National Heart, Lung and Blood Institute, the FDA and pharma to achieve these goals. A major early step of the Foundation was the formation of the COPD gene study under the direction of Dr. Edwin Silverman and Dr. James Crapo which involved the registration of 10,000 patients with COPD whose genetic status, pulmonary function and radiological status by computed tomography were characterized.

Under the present leadership of Dr. Byron Thomashaw and a dedicated Board of Directors, the COPD Foundation has instituted programs to assist patients with COPD in their immediate care, furthered the search for useful biomarkers of the disease, clarified comorbidities and increased public awareness of the disease internationally where

environmental pollutants in the home are etiological agents.

There is recognition that COPD clinically presents mixed patterns of disease with variations in the presence and severity of emphysema, bronchitis and the state of tissue inflammation. One of the major problems in understanding COPD is determining what mechanisms cause progression of COPD once initiating mechanisms such as exposure to tobacco smoke have ceased. Overall, the answer to this problem may await a deeper understanding of the genetic, cellular and molecular mechanisms that underlie the continued inflammatory state of the lung after initiating factors have been eliminated. Hopefully investigations into the injurious mechanisms, which may involve proteases and antiproteases, along with genetic predispositions, may progress in the coming years to give a clearer picture of the mechanisms, which determine the severity and progression of the disease and the resistance to treatment.

With the necessary financial support, the COPD Foundation can continue to be a leader in the effort to seek a cure, improve patient care and increase awareness.

Scott Cerreta, B.S., R.R.T:

Informing and educating individuals impacted by COPD has been a main goal of the COPD Foundation since it was established in 2004. There have been many changes in this department over the last 10 years, with an eye on continual growth well into the future.

In 2004, we started with educational materials for patients including The What's of COPD, The 1s, 2s and 3s of COPD and COPD fact sheets. In 2007 we introduced the Big Fat Reference Guide® (BFRG), a 400+ page manual for living well with COPD. Today, our educational materials are more robust with tools for health care professionals including the very popular Pocket Consultant Guide (PCG). The PCG is a guide for the health care professional to properly diagnose and treat COPD.

While comprehensive and a valuable reference, the BFRG is not practical for daily use. With this in mind, in 2010 the Slim Skinny Reference Guides® (SSRGs) were created. This is a series of the 10 most salient topics taken from the BFRG, pared down, and put into 12 to 16-page easy-to-read booklets. The SSRG series is great for patient support groups, pulmonary rehabilitation centers and inpatient hospital systems. In the last three years we have added a new booklet to the SSRG series, *COPD in the Hospital and the Transition Back to Home*. We also introduced the Disaster Preparedness Plan kit in 2013.

To increase the distribution of educational materials in 2011 we introduced the online catalogue. As always, individuals and family members impacted by COPD can continue to order free educational materials with free shipping by calling the COPD information line at 1-866-316-COPD (2673). In response to demand from health care professionals working in hospitals and clinics, we created a special ordering portal enabling organizations to distribute our materials directly to their patients. While these educational materials remain free, we do charge shipping fees to these organizations. Doing this helps the COPD Foundation to keep costs under control while continuing to make educational materials accessible to all those who need them. In 2011 we averaged 10 orders a month. Today, we average 20 orders a week.

As a national organization the COPD Foundation has been expanding our borders outside of the United States, working on an international presence since 2010. This led us to the translation of our three most common educational materials: the BFRG, SSRGs, and The 1's, 2s and 3's of COPD. This work was completed in 2013 and includes nine languages in addition to English: Arabic, Chinese, French, German, Hindi, Italian, Japanese, Portuguese and Spanish.

With the introduction of our new website in 2013, we added all of our educational materials for free download furthering easy access for patients and health care professionals free of charge. The COPD Foundation remains one of a few organizations continuing to provide educational materials at no cost. In addition to free downloads our website hosts an educational video library. Videos include COPD 101, a fascinating video tutorial on what happens to the lungs during COPD and why people are short of breath during simple activities, as well as inhaler training tutorials.

What about programs? The Pulmonary Education Program (PEP) is our most recent and successful. Launched in the fall of 2012 this program pre-packages COPD Foundation educational materials for partnering pulmonary rehabilitation centers to distribute to qualified COPD patients in their pulmonary rehabilitation programs. To date, we have over 210 pulmonary rehabilitation centers enrolled in PEP. Patients who graduate from a PEP pulmonary rehabilitation center may continue with support, encouragement and accountability by joining On Track with COPD. Pulmonary rehab graduates who join On Track receive a phone call once a month for 24 months from a trained infoline associate who provides encouragement and coaching to continue learning and staying healthy with COPD.

Over the past 10 years the COPD Foundation's education department has grown in many ways, but always with one mission in mind - to benefit and assist you, the individual impacted by COPD, to live as active, healthy and independent as possible. If you have not been to our website recently, be sure to check out the Educational Materials page under our "Learn More" category. There is a lot to see, and even more to come. www.copdfoundation.org

John W. Walsh:

Prior to 2004, there was no organization focused on COPD as a chronic condition that affected tens of millions of Americans and hundreds of millions of people worldwide. The focus was limited to clean air and tobacco control; COPD was highly stigmatized with the "shame and blame" of a self-inflicted disease. Individuals avoided the diagnosis, were embarrassed to be labeled with emphysema or chronic bronchitis and were given no hope of any recovery or any improved quality of life. There was no place to go for understandable, accurate and consistent information and there was very little public support for research and limited therapeutic options.

The COPD Foundation was founded in 2004 by individuals with COPD and leadership from the scientific and clinical community to focus exclusively on the mission to prevent and cure COPD and to improve the lives of all people affected by it. This unique partnership remains steadfast to this day and has expanded to include all stakeholders related to COPD. Without the dedication and commitment of these individuals and the seed funding and collaboration from the Alpha-1 Foundation, we would not have made the incredible progress that we have in the past 10 years.

Since inception, the COPD Foundation has maintained a strategic plan on how best to embrace and expand our three areas of focus: 1. Research 2. Education 3. Public policy and Advocacy. We have created the infrastructure to support the research community and accelerate targeted therapeutic development. We have established scientific credibility and created a balanced platform for federal agencies, academic medical research, community and industry to work together to support better diagnosis, improved treatment and development of new therapies to improve the quality of lives for individuals with COPD.

The COPD Foundation's active involvement and coordination of additional industry support for the COPDGene® Study gave us the ability to follow everyone longitudinally. Without question, the creation of this unique public-private partnership was a critical element in the renewal of funding from the National Heart, Lung and Blood Institute (NHLBI). This is the largest cohort ever studied and the largest grant ever awarded for lung disease, and the COPD Foundation is an integral part of its success under the leadership of Drs. James Crapo and Edwin Silverman.

The COPD Foundation was able to respond to the NHLBI's referral of interest about supporting bronchiectasis research by convening a group of experts and creating the Richard H. Scarborough Bronchiectasis Research Consortium and Registry. This has established the largest registry in the world of non-CF related bronchiectasis patients and networked numerous institutions focused on improving health outcomes and targeted therapies. It has also supported the impetus for bio-tech and pharmaceutical companies to initiate drug development for bronchiectasis in their pipeline.

When the Food and Drug Administration (FDA) needed a platform to take on the challenge of biomarker qualification for COPD, they came to the COPD Foundation. We convened a workshop on COPD biomarkers that resulted in a consensus recommendation

to organize a COPD Biomarker Qualification Consortium (CBQC) and the COPD Foundation coordinated the collaboration of five pharmaceutical companies, the NHLBI and several academic studies to create an integrated data base of up to 120,000 study subjects to support the qualification of biomarkers that should accelerate therapeutic development and approval.

Our CBQC currently has three biomarker qualifications in progress and identified the next three after convening the second workshop in 2013. This historic collaboration involving scientists from academia, industry and government would not have happened without the COPD Foundation.

There are numerous other examples of the progress we've made in research that we've reported on in the Digest and for which we will continue to keep the COPD community aware. Our immediate challenge and absolute priority is to create the largest registry of individuals with COPD ever assembled. We were thrilled to be awarded a Patient Centered Outcomes Research Institute (PCORI) Patient Powered Research Network (PPRN) contract to create a registry of 100,000 Americans with COPD over the next eighteen months. This may seem like a "mission impossible," but we're absolutely committed to proving this wrong and we are confident that the community will step up to the plate and join this incredible initiative. (Please call the C.O.P.D. Information Line at 866-316-COPD for information). Just imagine what we can accomplish with hundreds of thousands engaged in supporting our efforts.

Ten years ago there was barely any information available about COPD. Since then, the COPD Foundation has become an integral part of the NHLBI's *Learn More, Breathe Better Campaign*, establishing two key programs to support the campaign in 2007. The C.O.P.D. Information Line makes over 5,000 contacts a month, and all of the associates are fully trained individuals living with COPD or caregivers who provide the most direct resource ever for individuals affected by COPD. Our Mobile Spirometry Unit (MSU) generated awareness and has screened over 50,000 individuals across America. Our biggest impact with our MSU was the realization that you can test your lungs like you can test your blood pressure, cholesterol or blood sugar levels. Without the MSU, we would not have been able to conduct the Case Finding Validation Study. This study was recommended from our Case Finding Workshop we co-sponsored with the NHLBI. As a result of the findings and publication of this study, the COPD Foundation is now part of a grant from the NHLBI led by Dr. Fernando Martinez to create new, more accurate screeners and protocol to ensure the 12-14 million Americans who are symptomatic but not yet diagnosed with COPD can receive a proper diagnosis.

The DRIVE4COPD Campaign has created the momentum to vastly expand awareness of COPD across America. We have engaged corporate America with our Employer Toolkit and are using the data from the *COPD Uncovered Study* that reported a majority of those with COPD are still in the workforce. Imbedding our COPD message in corporate wellness programs and adding our risk screener to annual health risk assessment tools should help more people get diagnosed earlier. We've had more public exposure to COPD through the NASCAR collaboration than ever. To date, more than 3 million Americans have completed the five-question Risk Screener.

We have reported regularly about our public policy and advocacy initiatives in the *Digest* and many of you have signed on as state captains. We know that we can impact Congress; we know that we can impact access and support choice; and we have made great strides in all of this. And it's because of you, the COPD community. You've stepped up and made these things happen and beneficial change has come to the COPD Foundation and to all of you.

As I look back on the past 10 years, it's hard to believe that so much has been accomplished in a seemingly short amount of time. The resources, education, research, advocacy and community that exist now is overwhelming and thriving. We are so proud of everything the COPD Foundation has been able to do, and we look forward to the next 10 years and beyond.

We will continue to increase our resources, programs, educational materials, and events, and will work tirelessly to engage everyone in the COPD community. We welcome you to join our registry, become an advocate, and help us spread awareness for COPD.

To learn more, or to become involved in any capacity with the COPD Foundation, please visit our website at: www.copdfoundation.org or call (866) 316-COPD (2673).

EPOC: DE QUE SE TRATA

Gerard Turino, MD

Director Fundador, James P Mara Centro de Enfermedades Pulmonares

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Traducido por Sara G. Alegría, Ed.D.

Yo empecé mi carrera como médico asociado en los años cincuenta y tuve la suerte de recibir mi entrenamiento clínico inicial en la división universitaria del hospital Bellevue de Nueva York. Para entonces Bellevue era un centro de investigación cardiopulmonar donde los doctores Dickinson Richards y Andre Cournand, quienes con Werner Forsmann fueron los primeros en cauterizar el corazón humano en pacientes. Por este logro recibieron el Premio Nobel de Medicina y Fisiología en 1956.

En ese tiempo se podía caracterizar el conocimiento clínico de EPOC como indefinido. Lo que se conocía entonces era mayormente a través de patología anatómica, lo cual describía la presencia de enfisema en los pulmones de los pacientes que morían de enfermedad pulmonar. Esa descripción fue registrada primeramente por el famoso patólogo Francés Laennec a comienzos del siglo 19. Los bronquios se reconocieron como reducidos e inflamados y se observaron anormalidades en la estructura del tejido elástico a inicios de los años 60. En los 40 y a principios de los 50 se desarrollaron pruebas pulmonares de gran precisión. Pruebas de función pulmonar podían cuantificar la obstrucción del pasaje bronquial, la disminución de intercambio de aire y los cambios en las técnicas características del pulmón en términos de perdida de retroceso de pulmón. Sin embargo, la caracterización de EPOC como una entidad clínica en los pacientes variaba entre los clínicos.

"Debido a que definiciones y clasificaciones precisas no han estado disponibles se ha desarrollado una tendencia en trabajadores de disciplinas separadas a definir de modo diferente estas enfermedades por criterios seleccionados dentro de sus propios marcos de referencia o por sus métodos particulares de observación o técnicas de medición. La falta de definiciones y clasificaciones ampliamente aceptadas ha dañado la comunicación, lo cual, a su vez, ha retardado el progreso clínico e investigativo."

La siguiente declaración apareció en la introducción a una publicación sobre estándares de diagnóstico de la Sociedad Torácica Americana en 1962 titulada "Bronquitis Crónica, Asma y Enfisema Pulmonar".

En ese tiempo, en Inglaterra, la bronquitis crónica con su manifestación diaria de tos y esputo en ese medio se pensó que era el factor patogénico predominante de la enfermedad. En Holanda había énfasis en anormalidades alérgicas y en el papel de hiperreactividad de los conductos relacionado con el asma. En los Estados Unidos se reconocía que el enfisema anatómico era un elemento significativo en la enfermedad. El fumar, como factor causante, no fue aceptado como agente tóxico hasta los comienzos de los años sesenta.

Sin embargo, una mayor perspicacia ante los mecanismos de la enfermedad vino en 1963 cuando Laurell y Ericksson descubrieron en Suecia deficiencia de alfa-1 antitripsina como anormalidad genética asociada con el desarrollo de enfisema en hombres y mujeres a una temprana edad y a falta de exposición al humo del tabaco. La disminución de esta proteína en la sangre determinada genéticamente en dichos pacientes representó que una elastasa significativa, una enzima presente en neutrófilos (células blancas de la sangre) que químicamente degrada la elastina, no podía ser inhibida adecuadamente en el cuerpo. La elastina en el pulmón, que es una estructura esencial en los alveolos, sería degradada y la arquitectura alveolar destruida.

Una hipótesis de desequilibrio proteasa-antiproteasa tomó forma en este tiempo. Este mismo desequilibrio podría aplicarse también a pacientes con EPOC que tenían niveles normales de alfa-1 antitripsina en la sangre y tejido cuando se mostró que el aumento de oxidantes en fumadores podía desactivar la capacidad inhibitoria de la proteína alfa-1 antitripsina.

Para formalizar el enfoque clínico hacia EPOC, la Sociedad Torácica Americana convocó un taller sobre definiciones de EPOC en 1984. Este taller fue presidido por Gordon Snider y yo también fui un miembro. De este taller salieron definiciones que hoy aún se aplican. El enfisema se definió como un diagnóstico anatómico en el cual hubo destrucción de estructura alveolar, que se tenía que establecer por criterio anatómico o radiológico. Se definió la bronquitis como la presencia de tos y esputo casi todos los días por 3 meses cada año durante 2 años. La caracterización clínica de EPOC fue la presencia irreversible de obstrucción de los conductos con un FEV1 predicho inferior al 70%. A lo largo un tercer componente diagnóstico se añadió a la bronquitis crónica y enfisema bajo la sombrilla de EPOC que es el asma refractora reconociendo que algunos pacientes con asma persistente llegan a desarrollar obstrucción crónica de los conductos.

Escritos de posición significativa en el diagnóstico y tratamiento de EPOC fueron publicados por la Sociedad Torácica Americana en 1995 y actualizados en el 2004. También otros escritos de posición altamente significativa se publicaron con el nombre (traducido) "Estrategia Global para el Diagnóstico, Manejo y Presentación de Enfermedad Pulmonar Obstructiva Crónica," primeramente en el 2001 y revisados en el 2006. Esta estrategia de ORO, como se le llamó, formalizó el estado clínico de pacientes según determinación de pruebas de rendimiento de espirometría que incluyen mediciones de FEV1 y FEV1/FVC con graduaciones de enfermedad leve, moderada, severa y muy severa. El corte entre normal y EPOC, por el criterio ORO, es FEV1/FVC <70%. El criterio ORO ofreció la siguiente definición de EPOC:

La Enfermedad Pulmonar Obstructiva Crónica (EPOC) es una enfermedad prevenible y tratable con algunos efectos extra-pulmonares significativos. Su componente pulmonar se caracteriza por limitación de los conductos que no es completamente reversible. La limitación de los conductos es usualmente progresiva y asociada con una repuesta anormal inflamatoria del pulmón ante partículas nocivas o gases.

El desarrollo del "Criterio ORO" fue significativo en proveer un lenguaje común para clínicos que clasifican pacientes para el cuidado clínico y para proyectos de investigación.

Empezando hace 10 años se dieron cuenta que EPOC se estaba convirtiendo en un problema mayor de salud pública que estaba siendo ignorado por los pacientes y por los médicos ya que formas leves o moderadas de obstrucción de los conductos no eran reconocidos por los pacientes y atribuidos al aumento de peso, edad o desacondicionamiento. También hubo la realización de que EPOC debiera mejor ser diagnosticada en etapa inicial para eliminar la exposición al tabaco y tratar exacerbaciones.

John Walsh, él mismo un paciente de deficiencia de alfa-1 antitripsina y fundador de la Fundación Alfa-1, reconoció que una fundación nueva enfocada específicamente en EPOC, como un problema mayor de salud pública nacional e internacionalmente, pudiera servir a la medicina y al bien público. Por lo tanto la fundación EPOC se formó en el año 2002 con la misión de mejorar el cuidado de los pacientes, incrementar la conciencia pública, aumentar la educación con respecto a EPOC entre los médicos y demás personal y promover la investigación para incrementar el conocimiento de EPOC con el fin de lograr

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una cura. Desde sus comienzos la fundación EPOC trabajó en cooperación con la Sociedad Torácica Americana y el Instituto Nacional de la Salud para lograr estas metas. Un paso inicial mayor de la fundación fue la formación de un estudio genético bajo la dirección de los doctores Edwin Silverman y James Crapo que involucró el registro de 10,000 pacientes con EPOC cuyo estado genético, función pulmonar y estado radiológico por tomografía computarizada fueron caracterizados.

Bajo el presente liderazgo de Byron Thomashaw y la dedicación de la mesa directiva, la fundación EPOC ha instituido programas para asistir a pacientes con EPOC en su cuidado inmediato, promovido la búsqueda de biomarcadores útiles de la enfermedad, clarificado comorbilidades e incrementado la conciencia pública sobre la enfermedad internacionalmente donde contaminantes ambientales son agentes etiológicos.

Se reconoce que EPOC presenta clínicamente patrones de enfermedad mixtos con variaciones en la presencia y severidad de enfisema, bronquitis y el estado de inflamación de tejido. Uno de los mayores problemas para entender al EPOC es la determinación de que mecanismos causan progresión de EPOC una vez que mecanismos iniciadores como la exposición al humo del tabaco se han detenido. En conjunto, la respuesta a este problema pudiera aguardar un entendimiento más profundo de los mecanismos genéticos, celulares y moleculares que son la verdadera razón del continuado estado inflamatorio del pulmón luego de que los factores iniciales se han eliminado. Con suerte, las investigaciones sobre los mecanismos dañinos que pueden involucrar proteasas y antiproteasas, junto con predisposiciones genéticas, pueden avanzar en los años venideros para dar un cuadro más claro de los mecanismos que determinan la severidad y progresión de la enfermedad y la resistencia a tratamiento.

Con el apoyo financiero necesario la fundación EPOC puede continuar siendo un contribuyente mayor al esfuerzo para incrementar la conciencia pública del EPOC, mejorar el cuidado del paciente y buscar una cura.

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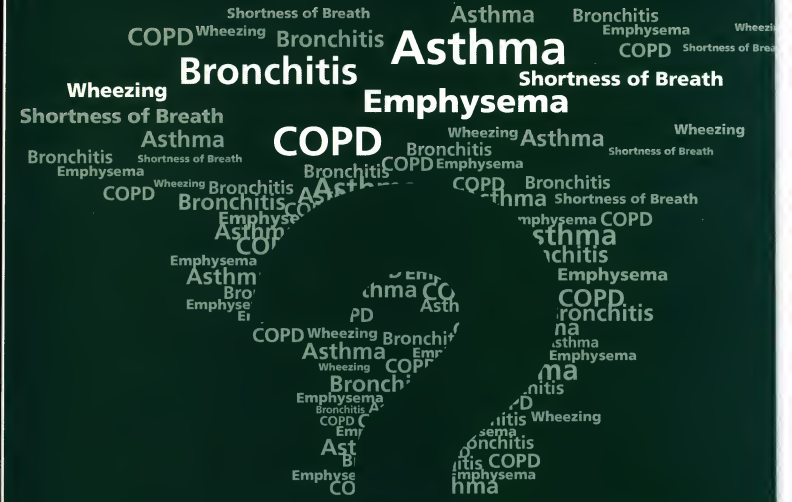

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